Amendments to the Specification:

Please replace the paragraph on page 16, line 15 with the following rewritten paragraph:

The antibody is appropriately from any source, including chicken and mammalian such as rodent, goat, primate, and human. Preferably, the antibody is from the same species as the species to be treated, and more preferably the antibody is humanized (i.e., has all human components) and the host is human. While the antibody can be a polyclonal or monoclonal antibody, preferably it is a monoclonal antibody, which can be prepared by conventional technology. The antibody is an IgG-1, -2, -3, or -4, IgE, IgA, IgM, IgD, or ~~an intraclass chimera~~ a chimeric antibody in which Fv or a CDR from one class is substituted into another class. The antibody may have an Fc domain capable of an effector function or may not be capable of binding complement or participating in ADCC.

Please replace the paragraph on page 16, line 27 with the following rewritten paragraph:

The term "immunosuppressive agent" as used herein for adjunct therapy refers to substances that act to suppress or mask the immune system of the host into which the graft is being transplanted. This would include substances that suppress cytokine production, downregulate or suppress self-antigen expression, or mask the MHC antigens. Examples of such agents include 2-amino-6-aryl-5-substituted pyrimidines (see U.S. Pat. No. 4,665,077, supra), azathioprine (or cyclophosphamide, if there is an adverse reaction to azathioprine); bromocryptine; glutaraldehyde (which masks the MHC antigens, as described in U.S. Pat. No. 4,120,649, supra); anti-idiotypic antibodies for MHC antigens and MHC fragments; cyclosporin A; steroids such as glucocorticosteroids, e.g., prednisone, methylprednisolone, and dexamethasone; cytokine or cytokine receptor antagonists including anti-interferon-γ, -β, or -α antibodies; anti-tumor necrosis factor-α antibodies; anti-tumor necrosis factor-β antibodies; anti-interleukin-2 antibodies and anti-IL-2 receptor antibodies; anti-L3T4 antibodies; heterologous anti-lymphocyte globulin; pan-T antibodies, preferably anti-CD3 or anti-CD4/CD4a antibodies; soluble peptide containing a LFA-3 binding domain (WO 90/08187 published 7/26/90); ; streptokinase; TGF-β; streptodornase; RNA or DNA from the host; FK506; RS-61443; deoxyspergualin; rapamycin; T-cell receptor (Cohen et al., U.S. Pat. No. 5,114,721); T-cell receptor fragments [Offner et al., Science, 251: 430-432 (1991)]; Howell, WO 90/11294; Ianeway, Nature, 341: 482 (1989); and Vandenbark, WO 91/01133]; and T cell receptor

antibodies (EP 340,109) such as T10B9. These agents are administered at the same time as or at separate times from the CD11a or CD18 antagonists ~~as~~ are used in this invention, and are used at the same dosages as or lesser dosages than as set forth in the art.

Please replace the paragraph on page 22, line 26 with the following rewritten paragraph:

The antagonist composition will be formulated, dosed, and administered in a fashion consistent with good medical practice. Factors for consideration in this context include the particular disorder being treated, the particular mammal being treated, the clinical condition of the individual patient, the cause of the disorder, the site of delivery of the agent, the method of administration, the scheduling of administration, and other factors known to medical practitioners. The "therapeutically effective amount" of LFA-1 antagonist to be administered will be governed by such considerations, and is the minimum amount necessary to prevent, ameliorate, or treat the LFA-1-mediated disorder, including treating rheumatoid arthritis, reducing ~~inflamatory~~ inflammatory responses, inducing tolerance of immunostimulants, preventing an immune response that would result in rejection of a graft by a host or vice-versa, or prolonging survival of a transplanted graft. Such amount is preferably below the amount that is toxic to the host or renders the host significantly more susceptible to infections.

Please replace the paragraph on page 36, line 10 with the following rewritten paragraph:

The flow cytometric studies (Fig. 5) showed an increase in percent splenic T cells expressing pan T, CD4, and CD8 markers. Furthermore, the percent of ~~LFP~~ LFA-1+ spleen cells did not differ substantially among M17-, IgG2a-, or non-treated mice. Moreover, there was no evidence of a decrease in yield of leukocytes per spleen in M17-treated mice compared to controls. Therefore, M17 does not appear to cause immunosuppression by central or peripheral lymphoid depletion. Furthermore, complete blood counts in these mice showed that M17 treatment did not suppress the number of lymphocytes compared to IgG2a-treated (n=3) control mice.

Please replace the paragraph on page 36, line 20 with the following rewritten paragraph:

Since treatment with M17 does not cause T cell depletion and since the results herein and those of others [Isobe *et al.*, *supra*~~)~~] show that LFA-1 is expressed after treatment with anti-LFA-1 MAbs, M17 may cause immunosuppression by functional inactivation of T cells. Thus, the function of immune cells from mice treated daily for two weeks with 4 mg/kg/day of M17 was assessed by determining the proliferative response of spleen cells to ConA *in vitro*. After 14 days of daily i.p. treatment with 4 mg/kg/day of either isotype control IgG2a (n=5) or M17 (n=5), spleens were removed and cells cultured in KC2000 (Hazelton Biologics, Lenexa, KS) in 96-well plates for 3 days with different concentrations of ConA (Vector, Burlingame, CA). Cell proliferation was assessed by pulsing with ^{3}H-TdR (ICN Radiochemicals, Irvine, CA, specific activity 6.7 Ci/mM) for 16-18 hours and the ^{3}H-TdR incorporation was determined by scintillation spectroscopy (Packard, Downers Grove, IL). For each ConA concentration, the mean disintegrations/min (dpm) was computed after subtracting background dpm for cells from control mice and M17-treated mice. The data were expressed as the percent change in dpm in spleen cells from M17-treated mice relative to dpm in spleen cells from control mice. $p>0.05$ determined using the Student's t test was considered not significant (NS).

Please replace the paragraph on page 41, line 6 with the following rewritten paragraph:

Part 2: Mixed Lymphocyte Response (MLR): One way human mixed lymphocyte cultures were established in 96-well flat-bottomed microtiter plates. Briefly, 1.5 x 105 responder PBMC in 200 µl of complete medium were co-cultured with an equal number of allogeneic irradiated (3000 rads) stimulator PBMC. Soluble ICAM-1 or anti-integrin antibodies [MHM24 (anti-CD11a) and H52 (anti-CD18), described in the references set forth above] were added at the initiation of cultures. Cultures were incubated at 37°C in 5% CO2 for 5 days, then pulsed with 1 µCi/well of 3H-thymidine (6.7 Ci/mmol, NEN, Boston, MA) for 16 hours. Cultures were harvested on a PHD cell harvester (Cambridge Technology, Inc., Watertown, MA). [^{3}H]TdR incorporation was measured with a ~~Beckman~~ BECKMAN™ brand scintillation counter (LS6800) and triplicate determinations were averaged. Data are expressed as net cpm. The mean [^{3}H]-TdR incorporated by control cultures was <1000 cpm.

Please replace the paragraph on page 42, line 8 with the following rewritten paragraph:

Part 3: CTL Killing Assay (A 4-hour ^{51}CR-release assay):

After 7 days of culture, CTLs (effector cells) were collected, washed three times, then adjusted to $1x10^7$ cells/ml. Target cells were collected and washed two times. Target cells were labeled with 150 μCi $Na^{51}CrO_4$ (5 mCi/ml: Amersham Corp., Arlington Heights, IL) for approximately 1 hour at 37°C, 5% CO_2 in air. Cells were washed four times, counted, and adjusted to 2 x 10^5 cells/ml. The CTL killing assay was set up in a ~~Corning~~ CORNING™ brand 96-well round-bottom plate. A total of 200 μl cells was added per well. 50 μl of target cells and 100 μl of effector cells at various concentrations, and 50 μl of antibodies [H52, anti-CD11b, anti-CD11a, anti-CD18, and anti-gp120 (7F11), which are all publicly available] at 500 ng/ml were added in triplicate to the plate.

Please replace the paragraph on page 43, line 21 with the following rewritten paragraph:

BALB/c mice (4-6 weeks old) obtained from Charles River were divided into four treatment ~~gruops~~ groups containing 6-8 mice per group. The mice were anesthetized i.p. with Ketamine/Xylazene/Acepromazine. A patch approximately 3x3 cm^2 was shaved on the abdomen of all the mice. A total of 50 μl of 10 mg/ml dinitrofluorobenzene (DNFB) was applied topically to the hair-free abdomen of the mice in groups 2-14. A ~~pipetman~~ PIPETMAN™ brand pipette was used to deliver the dose, enabling the wide end of the tip to be used to spread the DNFB over the skin.

Please replace the paragraph on page 44, line 4 with the following rewritten paragraph:

The mice were anesthetized with Metaphane™ brand anesthesia. With a ~~pipetmen~~ PIPETMAN™ brand pipette, 5 μl of ~~of~~ DNFB was applied topically to each side of the left pinnae of the mice in groups 2-14 (5 μl/side). The wide end of the ~~pipet~~ pipette tip was used to spread the DNFB over the ear. To each side of the right pinnae of the mice in groups 2-14 was applied topically 5 μl of the diluent of DNFB.